AMENDMENT TO THE TITLE

Please amend the Title of the Invention as follows:

~~HAIRLESS PROTEIN-INTERACTING PARTNER COMPLEXES AND METHODS THEREOF FOR THE BEAUTIFICATION AND/OR IMPROVEMENT OF MAMMALIAN SKIN~~ COMPOSITION COMPRISING A MOUSE HRt PROTEIN-HUMAN INTERACTING PARTNER PROTEIN COMPLEX

AMENDMENTS TO THE SPECIFICATION

Please amend the Specification as follows:

Please replace the paragraph at page 4, lines 17-25 with the following paragraph:

In yet another aspect of the present invention, a method of assaying a test compound for agonist or antagonist activity in the beautification and/or improvement of mammalian skin is disclosed. Said method comprises the steps of a) measuring a level of interaction between mouse HRt protein and the human interacting partner in the absence of the test compound; b) measuring a level of interaction between mouse ~~HR~~ HRt protein and the human interacting partner in the presence of the test compound; wherein when the level measured in step b) is greater than the level in step a), the test compound has agonist activity, and wherein when the level measured in step b) is less than the level in step a), the test compound has antagonist activity.

Please replace the paragraph at page 11, following the end notes of the table, from line 1 to page 12, lines 1-4, with the following paragraph:

References: Zlotogorski et al., (2002a), *Clinical and molecular diagnostic criteria of congenital atrichia with papular lesions*, J Invest Dermatol. 2002, 118:887-890. Ahmad et al (1999a), *Genomic organization of the humanhairless gene (HR) and identification of a mutation underlying congenital atrichia in the Arab Palestinian family*, Genomics 56, 141-148. Sprecher et al (1999b), *Atrichia with popular lesions resulting from a nonsense mutation within the human hairless gene*, J. Invest. Dermatol 113, 687-690. Ahmad et al (1999b), *A homozygous nonsense mutation in thezinc-finger domain of the human hairless gene underlines congenital atrichia*, J Invest Dermatol 113, 281-283. Ahmad et al (1998b), *A miss sense mutation in the zinc-finger domain of the human hairless gene underlines congenital atrichia in a family of Irish travelers*, Am J Hum Genet 63, 984-991. ~~Aitaet~~ Aita et al (2000), *A novel missense mutation (C622G) in the zinc-finger domain of the human hairless gene associated with congenital atrichia and popular lesions*, Exp Dermatol 9, 157-162. Kruse et al (1999), *Hairless mutations in two kindreds with autosomal recessive popular atrichia*, J Invest Dermatol 113, 954-959. Zlotogorski

et al (1998), *Congenital atrichia in five Arab Palestinian families resulting from a deletion mutation in the human hairless gene*, Hum Genet. 1998,103:400-404. Cichon et al (1998), *Cloning, genomicorganization, alternative transcripts and mutational analysis of the gene responsible for autosomal recessive universal congenital alopecia*, Hum Mol Genet 7, 1671-1679. Kruse et al (1999), *Hairless mutations in two kindreds with autosomal recessive ~~popular tarichia~~papular atrichia*, J Invest Dermatol 113, 954-959. Klein et al (2002), *A novel missense mutation affecting the human hairless thyroid receptor interaction domain 2 causes congenital atrichia*, J Invest Dermatol 119, 920-922. Ahmad et al (1998a), *Alopecia universalis associated with mutation in the human hairless gene*, Science 279, 720-724. Zlotogorski et al (2002b), *Evidence for pseudodominant inheritance of atrichia with papular lesions*, J Invest Dermatol 2002,118:881-886. Sprecher et al (1999a), *Identification of a genetic defect in the hairless gene in atrichia with popular lesions: evidence for phenotypic heterogeneity among inheritedatrichia*, Am J Hum Genet 64, 1323-1329, all of which are incorporated herein by reference.

Please replace the paragraph at page 17, lines 4-13, with the following paragraph:

In one aspect, a yeast interaction mating assay is employed, using two different types of host cells, strain-types a and alpha, of the yeast *Saccharomyces cerevisiae*. One set of host cells, for example the a strain cells, contains fusions of the HRt nucleotide sequences with the DNA-binding domain of a transcriptional activator, such as GAL4. The hybrid proteins expressed in this set of host cells are capable of recognizing the DNA-binding site on the reporter gene. The second set of yeast host cells, for example alpha strain ~~cells~~cells, contains nucleotide sequences as identified by the data provided in Table 1 fused to the activation domain of a transcriptional activator. In one embodiment, the fusion protein constructs are introduced into the host cell as a set of plasmids.

Please replace the paragraph bridging page 29, line 29 to page 30, lines 1-11, with the following paragraph:

Human Keratinocyte cDNA Library Screen for HRt interaction

Clontech Human Keratinocyte GAL4 cDNA library (complexity $2.5X10^6$) in pACT2 vector was ~~used~~ used to screen for HRt interacting proteins. A Leu^-, Trp^- yeast

strain with HIS3 and LACZ reporters for yeasts two hybrid interaction (strain L41) was first transformed with LexA BD-HRt ~~palsmid~~plasmid) for Trp^+ phenotype. The resulting L41/LexA BD-HR transformant was screened to ensure expression of LexA BD-HRt protein and also for lack of auto activation of the reporter genes HIS3 and LACZ. This strain was transformed with Keratinocyte AD-cDNA library and plated on $Trp^-Leu^-His^-$ plate to select for clones with positive interaction. Colonies that appeared on ~~selsctive~~ selective plates were subsequently tested for LACZ expression on X-GAL plates. A total of 90 colonies were identified. AD-plasmid was rescued from the 90 clones and retested for HRt bait ~~dependance~~dependence. Fifty four isolates were confirmed as positive. The positive clones are expected to contain an AD-IP (interacting partner) that interacts with BD-HRt. To characterize IP, the sequence of IP is determined and matched to the human genome database DNA sequence analysis and data base search for homology are performed using SeqWeb version 1.2 (in conjunction with Wisconsin Package Version 10.1).